of which will probably come to be recognized as playing important roles in the prevention of chronic diseases.

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See also: **Calcium**: Physiology. **Chromium**: Physiology, Dietary Sources and Requirements. **Electrolytes**: Water-Electrolyte Balance. **Energy**: Energy Requirements. **Folic Acid**: Physiology, Dietary Sources and Requirements. **Older People**: Physiological Changes. **Protein**: Requirements and Role in Diet. **Retinol**: Physiology. **Riboflavin**: Physiology. **Vitamin B_6**: Physiology.

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Physiological Changes

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Physiological changes in the elderly generally represent a decline in reserve capacity. They may be age-related without being an obligatory part of the ageing process. Nutritional factors may minimize these changes. These changes may, however, decrease nutritional tolerance for excesses or restrictions. These include decreased appetite, less taste appreciation and diminished handling of food in the gut, with alterations in motility and digestion. The hormonal responses to food, hormonal-like properties of foods themselves (such as phyto-oestrogens) and hormonal determinants of nutritional status (such as body composition), as well as body composition itself, are all valuable in the nutritional assessment and monitoring of the aged.

Body Composition

Body composition is dynamic throughout the life span. The changes that occur with ageing include a loss of height, muscle mass, bone mass and body water, while there is an increase in and redistribution of fat mass.

Body compartments

Body composition can be broadly classified into two sections: fat mass (FM) and fat-free mass (FFM). Fat mass incorporates all body lipids, while the fat-free mass includes all body constituents other than lipids such as minerals, proteins and water. Lean body mass (LBM) is similar to the FFM compartment except that it also includes essential lipids such as phospholipids. Finally, total body water (TBW), which accounts for most of the body's weight, includes both intracellular and extracellular water.

Physiological changes that occur with ageing

In both men and women height decreases with age. Alterations in posture, vertebral collapse, osteoporosis, loss of muscle tone and reductions in disc space all contribute to this loss of height. The rate of loss after maturity has been estimated to be between 0.5 and 2.0 cm per decade, with the loss being greater in older age.

Most literature indicates that peak bone density is achieved in the middle of the third decade, although it has been suggested that this may occur towards the end of adolescence. Involutional bone loss usually begins between the ages of 35 and 40 years, with the rate of bone loss varying according to the type of bone and the skeletal site. Overall women can lose at least half of their trabecular bone mass and about one-third of their cortical bone mass during their lifetime. For men this overall loss is less and represents about two-thirds of the loss experienced by women. Bone loss in accelerated in women at the menopause, but this rate slows about 5–10 years later.

There is decrease in FFM with ageing owing to a loss in muscle mass, bone mass and body organ mass. The extent of these losses depends on age itself, activity levels, gender (men lose lean body mass at a faster rate than women) and hormonal changes. By the seventh decade LBM has decreased by an average of 5 kg in women and 12 kg in men and skeletal muscle size has declined by as much as 40%. The kidney, liver and lungs become smaller with the ageing process. It has been estimated that the weight of the kidney is reduced by 9%, the liver by as much as 18% and the lungs by 11%.

As lean body mass decreases with advancing age, the percentage of body fat increases. There is a redistribution of subcutaneous fat from the limbs to the trunk and intra-abdominal fat increases. Total body water (TBW) in healthy elderly subjects also declines with age. This decline seems to parallel the decline in lean body mass, but whether the source of loss is from intracellular water or extracellular water remains unclear.

Measurements in the elderly

Change in body weight over time (in the absence of fluid imbalances) is one of the best indicators of changes in body composition. Limb length, unlike stature, is not altered by ageing and is therefore a useful surrogate for estimating original height. Two such measures include arm span and knee height. Estimating original height and comparing it with actual height may be a useful indicator of nutritional status, because the preservation of height may be looked upon as a positive sign in nutritional terms.

Skinfold thickness measurements of subcutaneous fat can be used to predict body fat content. In elderly subjects this method becomes less reliable because increased body fat is stored intra-abdominally and intramuscularly. The reliability of these measurements is further complicated by the decline in elasticity and compressibility of subcutaneous adipose in the elderly. Despite these limitations, serial skinfold measures are still useful in assessing changes in body composition.

Abdominal circumference is a useful indicator of abdominal obesity, although decreases in trunk length owing to conditions like osteoporosis may reflect some of the increase seen in abdominal circumference with ageing. Calf circumference is recognized by the World Health Organization to be the most sensitive measure of muscle mass in elderly.

Consequences of change

The change in body composition seen with ageing can be minimized with adequate exercise and nutrient intake, producing a more favourable outcome in terms of health. It has been shown the rate and degree of loss in LBM is an important predictor of length of survival and in some studies a higher body mass index has been associated with lower mortality in the elderly.

A reduction in muscle mass impacts on mobility, increases the risk of falls, and can adversely affect the functional capacity of other muscles such as the diaphragm. Loss of muscle mass also indicates a loss of protein reserves, which are drawn upon during episodes of illness. These losses increase the risk of malnutrition and immune dysfunction, conditions more prevalent among the aged.

Osteoporosis, a metabolic bone disease in which bone mass is reduced (either by decreased bone formation or by increased bone resorption, or both) causes fragility fractures. It accounts for a considerable proportion of the morbidity and mortality seen in ageing populations, for example the expected rate of survival in those sustaining a hip fracture is reduced by 12% or more.

The Gastrointestinal Tract

The mouth

Insofar as nutritional physiology is concerned the mouth and related structures contribute to taste and release of smells for the adjacent olfactory apparatus, mastication, a limited amount of starch digestion (by way of salivary amylase) and the initial phase of deglutination (swallowing).

Mastication may be impaired in the aged because of a reduction in masseter and medial pterygoid muscle mass with age, together with poorer movement control. Additionally the edentulous mouth may contribute to masticatory impairment, although retention of teeth to older age is improving and the need for associated dental and gingival care growing (osteoporosis of mandible and maxilla may contribute to gingival disease). Although dental health may influence food choice, nutritional status need not suffer if nutritious alternatives are sought. Salivary flow may be decreased in the aged, but in general it does not appear to be an inevitable accompaniment of ageing. More work is required on salivary composition in relation to age given its lubricating, buffering, remineralization, antimicrobial and digestive functions.

Oesophagus

Before a swallow reaches the oesophagus, its transit through the pharynx may be longer by up to 10% in over 65-year-olds. In the oesophagus itself, amplitude of contraction, extent of relaxation and smoothness of peristalsis may be reduced in the aged. Oesophageal motility disorders may be sufficient to be regarded as disease states like achalasia (absence of peristalsis) or diffuse oesophageal spasm. The term 'presbyoesophagus' is sometimes applied to oesophageal motility disorders in the aged. Although degenerative changes may occur as a consequence of ageing, these changes should not be accepted as part of the normal ageing process without further investigation. For instance, underlying conditions which may be responsible for motility changes include diabetes, strictures or malignant disease.

Stomach and duodenum

Decreased gastric function may supervene when gastritis affects the stomach, as in 'atrophic gastritis', in which a reduction in acid and also intrinsic factor (necessary for vitamin B_{12} absorption further down the gastrointestinal tract in the distal ileum) occurs, although this is usually not complete. It may have an autoimmune basis (usually in the gastric fundus, for example pernicious anaemia), but more commonly is attributable to long-standing infestation with *Helicobacter pylori* (usually in the gastric antrum), which can be eradicated. Crystalline vitamin B_{12} can be absorbed in elderly subjects with 'atrophic gastritis'.

While not a physiological process, the use of NSAIDS (nonsteroidal anti-inflammatory drugs) in the elderly can contribute to significant gastric and duodenal pathophysiology, notably haemorrhage and ulceration. At the same time, there is evidence that aspirin (acetyl salicylic acid, an NSAID) may protect against gastrointestinal cancers, raising interest that this may apply to salicylates occurring naturally in foods.

Pancreas

Pancreatic size decreases in later life, with duct dilation and parenchymal fibrosis. In the aged, volume, bicarbonate and enzyme output from the pancreas generally decline, and adaptation to dietary change is less adequate.

Small intestine

The absorption of carbohydrate, fat and protein appears to be less well tolerated in elderly people when loads are high. For instance, faecal fat and nitrogen excretion have been shown to increase more among aged adults compared with young adults when these nutrient loads are increased. Higher breath hydrogen excretion in elderly adults has also occurred in response to meals high in carbohydrate. Atrophic gastritis (mentioned above) produces a pH-dependent folic acid malabsorption in the jejunum in the aged and it can also contribute to calcium, iron and vitamin B_{12} malabsorption.

The bacterial microflora of the small bowel can also change and increase with age and contribute to malabsorption.

Liver

Liver mass and blood flow decrease in the aged, in part related to decline in lean body mass – but it may not be inevitable. Kupffer cells (liver reticuloendothelial cells) accumulate lipofuscin pigment. Protein synthesis generally declines and the responsiveness or regulation of albumin synthesis is less appropriate. Drug handling may diminish with implications for dosage.

Large bowel

Colon dysmotility and disordered defaecation contribute to constipation. Diverticula develop. Some of these alterations may reflect changes in diet, for example declining intakes of energy are often associated a reduced intake of dietary fibre and resistant starch.

The faecal microflora changes with a decrease in bifido-bacterium and increases in *Escherichia coli*, streptococcus, lactobacillus and *Clostridium perfringens*. Probiotics, like oligosaccharides and resistant starch, and probiotics (ingested live organisms), may have a place in minimizing these age-related changes.

Endocrine Function

There are several alterations in circulating hormone levels and action with advancing age; diseases often present in the elderly account for these changes, while other changes are due to ageing *per se*. There is an increased propensity for the aged to develop endocrine deficiency diseases (e.g. diabetes mellitus, hypothyroidism, hypogonadism) owing to a loss of functional reserve in many endocrine organs. The classic nonspecific presentation of endocrine disease in the aged is weight loss.

Insulin

Elderly people tend to have an impaired glucose tolerance compared with their younger counterparts owing to reduced insulin secretion and insulin sensitivity (cells lose their ability to respond to insulin because of defects in insulin receptors). The hyperglycaemia of ageing is more closely correlated with the increasing level of body fat and physical inactivity with advancing years than to the ageing process *per se*. Chromium has been suggested to have a role in normal glucose homeostasis because it forms part of the 'glucose tolerance factor' which promotes efficient insulin function. Chromium deficiency may take many years to develop and is more likely to occur in the elderly and has thus been implicated in the glucose intolerance of ageing. The main sources of chromium include brewer's yeast, blackstrap molasses, egg yolk, cheese, liver and wholegrain products.

Growth hormone and prolactin

Growth hormone levels also decline with advancing age and thus may explain a number of normal changes seen with ageing. These include the diminished nitrogen retention, the decrease in lean body mass, the increase in adipose tissue, and some of the osteopenia characteristically associated with ageing. The hormone prolactin (involved in lactation) also increases with age, and helps to maintain body fat in the elderly (and in breast-feeding mothers).

Thyroid hormones

There is a decreased production of thyroid hormones with ageing that is counterbalanced by a decreased thyroid hormone degradation. The prevalence of hypothyroidism and hyperthyroidism rises significantly in people aged over 60 years. Major changes in circulating thyroid hormones are mainly seen in the elderly when illness supervenes (e.g. euthyroid sick syndrome). These changes can mimic the changes seen with hypothyroidism. Malnourished elderly often present with the euthyroid sick syndrome. Hyperthyroidism is characterized by the hyperkinetic state, thyromegaly and eye signs, but in the elderly these signs are often replaced by heart failure, fatigue, depression, dementia and unexplained weight loss.

Aldosterone and dehydroepiandrosterone (DHEA)

The levels of aldosterone tend to fall with advancing age and thus tend to increase the propensity of older people to develop hyperkalaemia. Dehydroepiandrosterone (DHEA), a product made in the body during the synthesis of steroid hormones, declines dramatically with advancing age. Diminished levels of DHEA are associated with hypercholesterolaemia, hypertension and death from cardiovascular diseases in men. DHEA administration in animals prolongs life span, which may be related to its ability to reduce weight. DHEA deficiency results in reduced levels of nicotinamide adenine dinucleotide phosphate (NADP) and stimulation of lipogenesis, which has the potential to promote atherosclerosis. DHEA as a prescription drug is currently under investigation as it may have potential in reducing fat synthesis and enhancing fat utilization.

Testosterone and oestrogen

In the majority of older men there is a marked decrease in testosterone levels and a compensatory increase in luteinizing hormone (LH) and follicle-stimulating hormone (FSH) levels. In a number of elderly men, the hypothalamus fails to detect the decrease in testosterone adequately, resulting in the development of hypothalamic hypogonadism. Apart from the role of testosterone in maintaining normal sexual function, it also promotes nitrogen retention, maintains muscle mass, protects bone from excessive calcium loss and produces a feeling of wellbeing. The median age for the cessation of the menstrual cycle is 51 years (among women in the US) and is due to the failure of the ovaries to produce oestrogens and progesterone. As a consequence, the pituitary gland becomes more active and produces FSH and LH in greater quantity. Some ovarian secretion of oestrogen generally continues but gradually diminishes until it is inadequate to maintain the oestrogen-dependent tissues: the breasts and genital organs gradually atrophy; the decrease in protein anabolism

causes thinning of the skin and bones (oesteoporosis). Even after cessation of ovarian secretion of oestrogen, some oestrogen is found in plasma due to conversion of adrenally secreted androgens into oestrogen by nonovarian tissues. Androgens in the ageing female are responsible for hirsutism (e.g. hair on upper lip and chin) and abdominal fatness which in turn increases the risk of cardiovascular diseases and diabetes. Oestrogen favourably affects plasma low-density lipoprotein (LDL) cholesterol/high-density lipoprotein (HDL) cholesterol ratios, and this may explain why women have much less atherosclerosis than men until after the menopause, when the incidence becomes similar in both sexes. Consumption of naturally occurring oestrogens (phytoestrogens) in plant foods (especially legumes) have been shown to play a role in controlling menopausal symptoms and possibly in reducing the risk of breast and ovarian cancer by blocking oestrogen activity in cells, and by inhibiting endothelial cell proliferation and *in vitro* angiogenesis. The recognition that such foods (phytoestrogens) are effective indicates they may partially substitute for hormone replacement therapy (HRT).

Angiotensin and vasopressin

Elderly people are at increased risk of dehydration. This failure to develop an appropriate thirst response may be secondary to an impaired or inappropriate secretion of angiotensin and antidiuretic hormones, which may lead to hyponatraemia. Decreased free water clearance in response to vasopressin has also been noted, predominantly due to the age-related fall in glomerular filtration rate.

Activity Patterns

A moderate amount of physical activity (e.g. 30 min walk daily) throughout the life span is protective against early mortality, coronary heart disease, diabetes, stroke and osteoporosis. Exercise appears to affect the risk of death even more than heredity, smoking, hypertension or extremes in body weight. In addition to extending longevity, physical activity supports independence and mobility in later life by reducing the risks of falls and minimizing the risk of injury should a fall occur. Muscle mass and strength tend to decline with age, making older people vulnerable to falls and immobility. However, older adults who are active have less fat mass and more muscle mass, greater flexibility and better balance than those who are inactive.

Immobility

Stiffness, pain and declining mobility are unfortunately still perceived as stereotyped characteristics of 'normal ageing' rather than as being caused by potentially treatable conditions, e.g. adverse drug reactions, neurological, musculoskeletal and cardiovascular disorders or inappropriate bed rest. For example, phenothiazines are often prescribed for nonspecific 'dizziness' which is usually an undiagnosed gait disorder. These drugs can cause significant immobility by causing an extrapyramidal (parkinsonian) syndrome, postural hypotension and cognitive impairment. One dose is enough to cause immobility for weeks. Carers may unwittingly reinforce disability by discouraging activity, especially because of the fear of falls (**Table 1**). Once immobility is established ('bed and chair existence'), this will result in muscle weakness and wasting, poor balance, loss of endurance, loss of confidence, dependency and secondary complications of immobility (**Table 2**).

Physical activity

Adult energy needs decline by an estimated 5% per decade; basal metabolic rate is reduced in the aged by about 10–20% compared with their younger counterparts. This decline in metabolic rate reflects, by and large, the reduced lean body mass, which in

Table 1 Causes of gait disorder and falls in the aged which may ultimately lead to immobility

Neurological	**Musculoskeletal**
Stroke	Arthritis
Alzheimer's disease	Fractures
Parkinson's disease	Podiatric problems
Alcohol	Polymyalgia
Other cerebellar degenerations	Myopathy
Adverse drug reactions	**Cardiovascular**
Phenothiazines	Postural hypotension
Tricyclic antidepressants	Heart failure
Benzodiazepines	Angina
Antihypertensives	Claudication
Antiparkinson drugs	
Sensory	**Environmental**
Visions	Stairs
Hearing	Rugs
Fear of falling	Bathing, toileting
	Housework, shopping
Other	
Intercurrent illness	
Enforced dependency/bed rest	
Depression	
Pain	
Malnutrition	

Table 2 Some secondary complications of immobility

Venous thrombosis
Pulmonary embolism
Aspiration pneumonia
Orthostatic hypotension
Leg oedema
Impaired glucose tolerance
Constipation
Urinary incontinence
Increased bone fracture risk
Pressure sores/ulcers

turn requires that older adults eat less food energy to maintain their weight. It has been suggested that activities of growth hormone and testosterone, which promote lean tissue growth, are reduced with ageing; this may contribute to the shift in balance from lean to adipose tissue with age. A decreased capacity for muscle fibre regeneration and a decreased trophic effect of the autonomic nervous system on muscle may also be partly responsible. However, regular exercise not only impedes this loss of lean body mass, but also increases energy expenditure and energy intake, thereby enhancing nutrient intakes. Age-related conditions such as cardiovascular disease, musculoskeletal disease, osteopenia, obesity and others can result in a decline in physical activity. There is also a decline in physical working capacity (Vo_2max), amounting to about 10% per decade between ages 25 and 65 years. This means that the same physical tasks require a greater physical effort in the aged. Physical training can correct these age-related changes in physical working capacity by as much as 50%.

Appetite and Taste

Taste

The sensation of taste is perceived by taste buds located on the tongue, roof of the mouth, pharynx, larynx and the upper third of the oesophagus. Taste buds are situated in small protrusions on the surface of the tongue called papillae. Each taste bud is innervated by three cranial nerves and consists of a number of sensory receptor cells surrounded by supporting cells. These cells are renewed every 10–10.5 days. Chemical substances pass through a pore at the surface of the taste bud to reach the taste receptors. Finally, interpretation of taste occurs in the taste centre of the cerebral cortex.

Losses in taste sensitivity (hypogeusia) occurs in most elderly people. This loss was initially thought to be due to a reduced number of taste buds, but recent data indicate that a decline in the number of receptors may be responsible. The ability to detect a taste (threshold) is elevated in the aged, but the threshold varies with different substances. Taste thresholds on average tend to be 2–2.5 times higher in elderly adults when compared with young adults. For some substances, however, the threshold may be more than 20 times higher in the elderly.

Smell

The sense of taste is closely related to the sense of smell. In order to smell a chemical substance it must come into contact with olfactory receptors that are located within the olfactory mucosa at the roof of the nasal cavity. These olfactory receptors are bipolar sensory neurons that possess cilia at one end and axons at the other end. The cilia which arise from the end of the neuron facing the surface of the nasal cavity are thought to be stimulated by chemical substances. The axons of these bipolar neurons unite to form bundles which pass through a small bone known as the cribriform plate to reach the olfactory bulbs. The olfactory bulbs that are situated in the anterior section of the brain contain groups of cells called glomeruli. With ageing many of these cells degenerate. The axons of the bipolar neurons (which reach the olfactory bulbs) synapse with the dendrites of other neurons. The axons from these neurons form the olfactory tract which conveys impulses to the 'old brain' for processing.

Olfaction usually begins to decline during the sixth decade and deteriorates further after the seventh decade. The threshold for the sense of smell is higher and the ability to discriminate between different odours is diminished in the elderly. This diminished sense of smell may result, in part, from the degeneration of cells in the olfactory bulbs or in the neurons found in the 'old brain'. Neurons found in the 'old brain' (which consists of the hippocampus, amygdala and the prepyriform cortex) are the first neurons to show signs of degeneration with ageing.

Appetite

Factors which control food intake are complex and not fully understood. Salivation, gastric secretions and hormonal stimulation can arise from the sight, smell, taste and thought of food. There are 10 gastrointestinal hormones which have been shown to inhibit food intake. These include cholecystokinin, bombesin, gastrin, secretin, glucagon, insulin, somatostatin, neurotensin, substance P and pancreatic peptide. Neurotransmitters such as serotonin, norepinephrine and the opiates are known to influence food choice, however the exact mechanisms by which these operate is not clear. Changes that occur to these hormones or neurotransmitters and their

effect on appetite in the elderly need further investigation.

The homeostatic mechanisms that control appetite may be impaired in the elderly. Several studies have shown elderly men less capable of regulating their energy intakes after dietary manipulations compared with younger adults. Other studies report a drop in energy intakes with ageing. This reduction can be explained, in part, by the age-related drop in basal metabolic rate. However, the decline in physical activity (and presumably appetite) that accompanies ageing appears to be the major contributor to the fall in energy intakes.

See also: **Cancer**: Epidemiology of Breast Cancer. **Dehydration**: Physiological Effects and Management. **Exercise**: Beneficial Effects. **Gastrointestinal Tract**: Structure and Function of the Stomach; Structure and Function of the Small Intestine; Structure and Function of the Colon. **Osteoporosis**: Aetiology.

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Nutritionally Related Problems

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The population over the age of 55 years is increasing rapidly all over the world. In industrialized countries the proportion of elderly people will increase by